History

A 4-year-old 31.0-kg (68.2-lb) castrated male mixed-breed dog was examined by the cardiology service at the Colorado State University Veterinary Teaching Hospital for a scheduled recheck examination and pacemaker interrogation. Previous diagnoses include bartonellosis; severe, chronic hepatitis; and third-degree (ie, complete) atrioventricular (AV) block. Intermittent atrial standstill attributable to some degree of sinoatrial dysfunction was also present. Two weeks prior to the recheck examination, a pacemaker with transdiaphragmatic epicardial leads had been placed in response to a 3-month history of labored breathing, lethargy, and syncope. Epicardial leads had been selected, rather than transvenous, endocardial leads, because the previously diagnosed bartonellosis raised concerns that there would be a greater chance for infection-associated lead dislodgement with endocardial leads. One week prior to the recheck examination, inappetence and a decrease in energy level were noted along with occasional episodes of transient, mild tachypnea.

On physical examination, the dog was bright, alert, and responsive. Mucous membranes were pink and moist, with a capillary refill time of 2 seconds. Heart rhythm was regular; heart rate was 60 beats/min. A grade III of VI left apical systolic heart murmur was heard during auscultation. Femoral pulses were strong and synchronous. Lung field sounds were unremarkable, with normal bronchovesicular sounds heard bilaterally. Results of a CBC and serum biochemical profile were unremarkable, other than moderate thrombocytopenia (100.0 X 10^3 platelets/µL; reference range, 200.0 X 10^3 platelets/µL to 500 X 10^3 platelets/µL) and mild hyperbilirubinemia (0.4 mg/dL; reference range, 0.0 to 0.3 mg/dL). Pacemaker interrogation revealed a complete failure to capture regardless of amplitude and pulse width settings. Electrocardiography revealed an underlying sinus rhythm with intermittent atrial standstill and third-degree AV block with regularly occurring junctional escape complexes at a rate of 60 to 65 beats/min. Thoracic radiography revealed dislodgement of at least 1 of the 2 epicardial leads from the left ventricular apex (Figure 1).

A ventral celiotomy and transdiaphragmatic approach were planned to replace the epicardial pacemaker leads. An American Society of Anesthesiologists status of IV was assigned on the basis of the concurrent severe, chronic hepatitis and third-degree AV block with sinoatrial dysfunction.

The patient was premedicated with morphine (0.7 mg/kg [0.32 mg/lb], SC) and atropine (0.03 mg/kg [0.014 mg/lb], SC). Transthoracic pacemaker leads were attached to the patient’s thoracic wall in case temporary pacing was required while the dog was anesthetized. Anesthesia was induced with midazolam (0.48 mg/kg [0.22 mg/lb], IV) and etomidate (0.39 mg/kg [0.18 mg/lb], IV). A 12-mm (internal diameter) cuffed orotracheal tube was inserted, and the patient was connected to a circle system anesthetic circuit. Lactated Ringer’s solution was administered (10 mL/kg/h [4.5 mL/lb/h], IV, for the first hour; then 5 mL/kg/h [2.3 mL/lb/h], IV, thereafter) throughout the surgical procedure. Anesthesia was maintained with isoflurane in oxygen (initial isoflurane vaporizer setting, 1.5%; initial oxygen flow rate, 2 L/min) and a constant-rate infusion of fentanyl (10 µg/kg/h, IV).

Figure 1—Ventrodorsal radiographic view of the thorax of a dog examined because of lethargy, inappetence, and tachypnea of 1 week’s duration. A pacemaker with transdiaphragmatic epicardial leads had been implanted the week before. Notice that neither of the pacemaker leads appears to be associated with the epicardial surface of the heart.

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The following variables were monitored with a multiparameter anesthesia monitor: oxygen saturation measured by means of pulse oximetry (SpO₂), end-tidal partial pressure of carbon dioxide (PetCO₂), and rectal temperature. An arterial catheter was inserted in the dorsal pedal artery in the right hind limb and attached to a fluid-filled line and transducer. The transducer was positioned at the level of the heart to allow continuous, direct measurement of blood pressure. A lead II ECG was monitored continuously for changes in heart rate and rhythm.

Immediately after anesthesia was induced, SpO₂ was 98%, PetCO₂ was 42 mm Hg, mean arterial blood pressure was 105 mm Hg, rectal temperature was 37.6°C (99.8°F), respiratory rate was 30 breaths/min, and ventricular heart rate was 75 to 85 beats/min. Examination of the lead II ECG revealed an underlying sinus rhythm with sinoatrial dysfunction and third-degree AV block with a junctional escape rhythm. Within 10 minutes after the dog was anesthetized, the ventricular heart rate decreased to 40 to 45 beats/min and the mean arterial blood pressure decreased to 52 mm Hg, whereas delivered isoflurane concentration and all other vital signs remained unchanged (Figure 2).

A constant-rate infusion of dopamine (10 µg/kg/min, IV) was started but had no discernible effect on heart rate or blood pressure. While the dopamine infusion was continued, a constant-rate infusion of dobutamine (10 µg/kg/min, IV) was also started 15 minutes after initiation of the dopamine infusion. Changes in vital signs observed within 2 minutes after the start of the dobutamine infusion consisted of an increase in ventricular heart rate to 80 to 85 beats/min and an increase in mean arterial pressure to 75 to 80 mm Hg (Figure 3).

The atrial rate was unchanged during infusion of dopamine and dobutamine. When the dosage of dobutamine was decreased to 5 µg/kg/min, IV, examination of the lead II ECG revealed that the patient’s electrical heart rhythm alternated between 2 distinct morphologies of junctional escape complexes and more wide and bizarre ventricular escape complexes. Whenever the constant-rate infusion of dobutamine was stopped, the ventricular heart rate returned to 40 to 45 beats/min, the heart rhythm returned to the ventricular escape morphology, and the mean arterial blood pressure decreased to 50 to 55 mm Hg. This phenomenon was repeatable throughout the anesthetic period, prior to replacement of the epicardial pacemaker leads. In addition, different rates of dobutamine infusion (5 and 10 g/kg/min) reliably and repeatedly produced the aforementioned effects on heart rate, mean arterial blood pressure, and the lead II ECG rhythm morphologies, whereas the delivered isoflurane concentration and all other vital signs remained unchanged.

The epicardial pacemaker leads were successfully replaced, and the dog recovered without complications.

**Question**

What is the best option to treat dogs with third-degree AV block? What options should be considered when anesthetizing dogs with third-degree AV block?

**Answer**

In dogs with third-degree AV block, the mainstay of treatment is permanent pacemaker placement during general anesthesia. Owing to poor cardiac output in dogs with third-degree AV block, several temporary...
methods to support cardiac output during induction and maintenance of general anesthesia have been described. The goals of these temporary treatments are to support heart rate, myocardial contractility, and cardiac output until the permanent pacemaker is placed. Methods that have been described to support cardiac output in anesthetized dogs with third-degree AV block prior to permanent pacemaker implantation include medical treatments, such as isoproterenol, epinephrine, atropine, dopamine, and dobutamine, and nonmedical treatments, such as transesophageal pacing, transthoracic pacing, and temporary transvenous pacemaker placement. In the dog described in the present report, atropine was used as part of the premedication protocol. In addition, after induction of anesthesia, constant-rate infusions of dopamine and dobutamine were administered as described.

Discussion

Several medical options have been described for managing third-degree AV block in humans, including theophylline, epinephrine, ephedrine, and isoproterenol. It is believed that the use of isoproterenol or epinephrine can cause an alteration in the foci of the ventricular beats within the specialized conduction system so that they are nearer to the AV nodal tissue. In dogs with third-degree AV block, atropine is used to elicit an improvement in heart rate and AV conduction. However, improvements in AV nodal conduction are rarely seen. Isoproterenol has reportedly been successfully used to treat some dogs with second-degree and third-degree AV block by increasing heart rate and altering ventricular activity to normal sinus rhythm.

The current standard of care for treatment of third-degree AV block in dogs is widely considered to be permanent pacemaker placement. Because permanent pacemaker implantation requires general anesthesia, regardless of whether transvenous endocardial or epicardial leads are used, various temporary cardiac pacing methods that can support cardiac output while the permanent pacemaker implantation procedure is in progress have been developed. In particular, transvenous placement of a temporary pacemaker is a common method of temporary cardiac pacing. However, this requires real-time imaging, such as fluoroscopy, along with sufficient technical experience to perform the procedure quickly and efficiently. For this reason, several less invasive methods have been developed for providing temporary cardiac pacing. Transthoracic cardiac pacing can be used for temporary cardiac pacing in dogs, and this method does not result in clinically important myocardial injury. However, it does cause substantial skeletal muscle stimulus and pain in conscious dogs and therefore should only be used following induction of general anesthesia. Transesophageal and transgastric cardiac pacing have been successfully used for atrial and ventricular pacing, respectively, in humans with bradyarrhythmias, but in dogs, only transesophageal atrial pacing has reportedly been successful.

Atropine, a parasympatholytic drug, was used for premedication of the dog described in the present report. With AV nodal block, there exists some abnormality preventing proper electrical conduction from the SA node to the ventricular myocardium. In dogs with first-degree or second-degree Mobitz type I AV block, the lack of proper electrical conduction through the AV node is typically a result of increased parasympathetic tone, which causes an increase in the dispersion and relative degree of refactoriness in AV nodal tissue. Following administration of atropine in these dogs, AV nodal conduction should return to normal, as evidenced by resolution of the conduction abnormalities and an increase in heart rate to ≥ 180 beats/min. Second-degree Mobitz type II and third-degree AV block are most often caused by organic AV nodal disease and typically do not respond to atropine administration. As expected, no improvement in AV nodal conduction or heart rate was seen following atropine administration in the dog described in the present report. In fact, sinoatrial dysfunction became apparent when no change in atrial rate occurred after atropine administration. This was the reason why additional atropine was not given when the dog developed bradycardia following induction of anesthesia. Morphine, a pure μ-opioid receptor agonist, was also used as part of the premedication protocol for its analgesic and mild sedative properties. Mild sedation is desirable to prevent excitement during anesthetic induction and allow lower doses of anesthetic agents to be used. To our knowledge, there have been no reports that morphine can affect AV nodal conduction. However, it is known that opioids can have negative inotropic effects. Although morphine's negative inotropic effects typically are not important in dogs with normal cardiovascular function, they may be in dogs with systolic dysfunction. However, given the transthoracic cardiac pacing in this dog, the disadvantages of morphine's negative inotropic effects were thought to be outweighed by the advantages of its sedative effects for anesthetic induction. Acepromazine, a phenothiazine derivative, should be avoided in animals with severe hepatic or cardiac disease. Although acepromazine can be used as a preanesthetic sedative in some dogs, it requires extensive hepatic metabolism for excretion, can substantially decrease systemic blood pressure, and has a long half-life, and its effects are not reversible.

Etomidate, an ultrashort-acting injectable anesthetic, was used for anesthetic induction in the dog described in the present report because of its rapid effects and because it causes only mild cardiovascular depression and is not arrhythmogenic. However, etomidate is relatively expensive, causes pain on IV injection, can potentially induce mild to moderate myoclonus or emesis, and suppresses the hypothalamic-pituitary-adrenocortical axis for several hours. Midazolam, a benzodiazepine, was also used for anesthetic induction because of its sedative effects, its ability to minimize the dose of etomidate required, and its ability to prevent etomidate-induced emesis and myoclonus. Use of a combination of etomidate and midazolam is the authors’ preferred method for anesthetic induction in dogs with clinically important cardiovascular compromise.

Drugs used to manage third-degree AV block in anesthetized dogs include positive inotropic agents.
such as isoproterenol, epinephrine, dopamine, and dobutamine. Positive inotropic agents are desired for anesthetized patients with third-degree AV block because these patients may have poor systolic function owing to chronically increased ventricular blood volume. In addition, these dogs would be more severely affected by the negative chronotropic, negative inotropic, and vasodilatory effects associated with inhalant anesthetics. In this dog, a constant-rate infusion of fentanyl allowed us to decrease the concentration of isoflurane required to maintain anesthesia, thus decreasing the negative effects of isoflurane on cardiac output and vascular resistance.12 Isoproterenol has been used to augment cardiac output during anesthesia and produces strong activation of the β1-adrenergic receptors.13 However, in our experience, the vasodilatory effects of isoproterenol in anesthetized dogs regularly cause an unacceptable decrease in blood pressure. In addition, isoproterenol can cause a large proportion of the cardiac output to be diverted to the skeletal muscles, which are rich in β2-adrenergic receptors, and this may adversely affect perfusion of other vital organs.13,14 Epinephrine can also be used to increase heart rate, but its arrhythmogenic properties make it an undesirable option for use in anesthetized dogs.

The chronotropic, inotropic, and dromotropic effects of sympathomimetic drugs such as dopamine and dobutamine have been evaluated in dogs with third-degree AV block. Dopamine is a norepinephrine precursor that causes the release of norepinephrine and stimulates β1-adrenergic receptors.15 Dopamine has been shown to increase the automaticity, conduction velocity, and rate of diastolic depolarization of Purkinje fibers.15 In low cardiac-output states such as seen in anesthetized dogs with third-degree AV block, the myocardial fibers can become depolarized owing to extreme myocardial fiber stretch secondary to the abnormal increase in ventricular blood volume. Dopamine promotes the repolarization of these myocardial fibers so that they are better able to respond to external electrical stimuli such as cardiac pacing.15 In our experience, administration of dopamine during anesthetic procedures in dogs with third-degree AV block can cause hypertension without affecting the heart rate. Dobutamine is a synthetic derivative of isoproterenol that strongly activates β1-adrenergic receptors and only weakly activates β2-adrenergic receptors.13,14 Dobutamine has been shown to increase sinoatrial node automaticity, AV nodal conduction rate, overall heart rate, aortic blood flow, and systemic blood pressure.13–18 In humans and dogs with healthy and ischemic myocardial tissue, dobutamine has been shown to cause atrial arrhythmias, reduce ventricular myocyte refractoriness, and precipitate ventricular arrhythmias.13,19 Patients with preexisting heart disease or a history of arrhythmias are more likely to develop ventricular arrhythmias during dobutamine infusion.15 Currently, dobutamine is commonly used in human medicine to pharmacologically imitate exercise-induced myocardial stress during stress echocardiography.20,21 In veterinary medicine, dobutamine is primarily used to support cardiac output and maintain adequate blood pressure in anesthetized patients, including dogs.22,23 However, to our knowledge, the use of dobutamine in dogs with third-degree AV block to successfully increase ventricular rate and blood pressure without complications such as arrhythmias, increased atrial rate, hypertension, or hypotension has not been reported previously. In addition to the dog described in the present report, we successfully managed a second anesthetized dog with third-degree AV block using a combination of dobutamine and dopamine. Both of these dogs responded to combined dopamine-dobutamine administration with an increase in ventricular heart rate of 10 to 30 beats/min and an increase in mean arterial blood pressure of 15 to 20 mm Hg.

It has been shown that dogs with chronic third-degree AV block are at greater risk for developing ventricular arrhythmias when anesthetized than when they are conscious.15–19,24 Although there are some conflicting reports as to whether dopamine or dobutamine is more arrhythmogenic, it is generally accepted that both drugs possess an inherent ability to promote or cause ventricular arrhythmias in dogs. It is unclear why dobutamine alone did not affect the blood pressure in the dog described in the present report. It is possible that the dose was not high enough for this particular dog or that an effect may have been observed if a longer period had elapsed prior to initiation of the dobutamine infusion. Dopamine and dobutamine were used simultaneously not only to address hypotension and systolic function but also with the thought that by combining these 2 drugs, an increase in ventricular activity and cardiac output may be elicited. It is unclear whether the change in ventricular focus alone caused the increase in systemic blood pressure in this patient or whether blood pressure would have increased with infusion of a combination of dopamine and dobutamine regardless of ventricular rate. The etiology of the cardiac disease in this dog was unclear, so it is unknown whether all dogs with third-degree AV block undergoing anesthesia would respond similarly. To the authors’ knowledge, there are no current reports of dogs with third-degree AV block responding to infusion of dobutamine or dopamine, individually or in combination, in the manner described in this report. However, given our findings in this dog, it is possible that infusion of a combination of dopamine and dobutamine may be a valuable method to maintain adequate ventricular heart rate and systemic blood pressure in some dogs with chronic third-degree AV block undergoing anesthetic procedures.

References